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                   (ROSPATENT) added to list of core patent offices covered
 NEWS  4  FEB 28   PATDPAFULL - New display fields provide for legal status
                   data from INPADOC
 NEWS  5  FEB 28   BABS - Current-awareness alerts (SDIs) available
 NEWS  6  FEB 28   MEDLINE/LMEDLINE reloaded
 NEWS  7  MAR 02   GBFULL: New full-text patent database on STN
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 NEWS  9  MAR 03   MEDLINE file segment of TOXCENTER reloaded
 NEWS  10 MAR 22   KOREAPAT now updated monthly; patent information enhanced
 NEWS  11 MAR 22   Original IDE display format returns to REGISTRY/ZREGISTRY
 NEWS  12 MAR 22   PATDPASPC - New patent database available
 NEWS  13 MAR 22   REGISTRY/ZREGISTRY enhanced with experimental property tags
 NEWS  14 APR 04   EPFULL enhanced with additional patent information and new
                   fields
 NEWS  15 APR 04   EMBASE - Database reloaded and enhanced
 NEWS  16 APR 18   New CAS Information Use Policies available online
 NEWS  17 APR 25   Patent searching, including current-awareness alerts (SDIs),
                   based on application date in CA/CAplus and USPATFULL/USPAT2
                   may be affected by a change in filing date for U.S.
                   applications.
 NEWS  18 APR 28   Improved searching of U.S. Patent Classifications for
                   U.S. patent records in CA/CAplus

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       BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
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          0*  FILE ADISNEWS
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          0*  FILE AQUALINE
          0*  FILE BIOCOMMERCE
          0*  FILE BIOENG
         15   FILE BIOSIS
          1*  FILE BIOTECHABS
          1*  FILE BIOTECHDS
          4*  FILE BIOTECHNO
         14   FILE CAPLUS
          0*  FILE CEABA-VTB
          0*  FILE CIN
          5   FILE DGENE
         10   FILE EMBASE
         10*  FILE ESBIOBASE
          0*  FILE FEDRIP
          0*  FILE FOMAD
          0*  FILE FOREGE
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          6   FILE LIFESCI
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         10   FILE MEDLINE
          0*  FILE NTIS
          0*  FILE NUTRACEUT
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          5   FILE TOXCENTER
         11   FILE USPATFULL
          1   FILE USPAT2
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L1    QUE FODRIN(P) GLUTAMATE

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                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   1.18       1.60

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FILE 'SCISEARCH' ENTERED AT 13:54:26 ON 03 MAY 2005
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L2 47 L1

=> dup rem l2
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L3 19 DUP REM L2 (28 DUPLICATES REMOVED)

=> d bib, hit 1-

YOU HAVE REQUESTED DATA FROM 19 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 19 MEDLINE on STN DUPLICATE 1
AN 2005046321 MEDLINE
DN PubMed ID: 15673434
TI A novel scaffold protein, TANC, possibly a rat homolog of Drosophila rolling pebbles (rols), forms a multiprotein complex with various postsynaptic density proteins.
CM Erratum in: Eur J Neurosci. 2005 Feb;21(3):825. Usada, Nobuteru [corrected to Usuda, Nobuteru]
AU Suzuki Tatsuo; Li Weidong; Zhang Jing-Ping; Tian Qing-Bao; Sakagami Hiroyuki; Usuda Nobuteru; Usada Nobuteru; Kondo Hisatake; Fujii Toshihiro; Endo Shogo
CS Department of Neuroplasticity, Institute on Ageing and Adaptation, Shinshu University Graduate School of Medicine, 3-1-1 Asahi, Matsumoto 390-8621, Japan.. suzukit@sch.md.shinshu-u.ac.jp
SO European journal of neuroscience, (2005 Jan) 21 (2) 339-50.
Journal code: 8918110. ISSN: 0953-816X.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AB098072
EM 200504
ED Entered STN: 20050128
Last Updated on STN: 20050429
Entered Medline: 20050428
AB We cloned from the rat brain a novel gene, tanc (GenBank Accession Number AB098072), which encoded a protein containing three tetratricopeptide repeats (TPRs), ten ankyrin repeats and a coiled-coil region, and is possibly a rat homolog of Drosophila rolling pebbles (rols). The tanc gene was expressed widely in the adult rat brain. Subcellular distribution, immunohistochemical study of the brain and immunocytochemical studies of cultured neuronal cells indicated the postsynaptic localization of TANC protein of 200 kDa. Pull-down experiments showed that TANC protein bound PSD-95, SAP97, and Homer via its C-terminal PDZ-binding motif, -ESNV, and **fodrin** via both its ankyrin repeats and the TPRs together with the coiled-coil domain. TANC also bound the alpha subunit of Ca2+/calmodulin-dependent protein kinase II. An immunoprecipitation study showed TANC association with various postsynaptic proteins, including guanylate kinase-associated protein (GKAP), alpha-internexin, and N-methyl-D-aspartate (NMDA)-type **glutamate** receptor 2B and AMPA-type **glutamate** receptor (GluR1) subunits. These results suggest that TANC protein may work as a postsynaptic scaffold component by forming a multiprotein complex with various postsynaptic density proteins.

L3 ANSWER 2 OF 19 SCISEARCH on STN
AN 2004:953753 SCISEARCH
GA The Genuine Article (R) Number: 864WX
TI Ischemia-induced increase in long-term potentiation is warded off by specific calpain inhibitor PD 150606
AU Farkas B; Tantos A; Schlett K; Vilagi I (Reprint); Friedrich P

CS Lorand Eotvos Univ, Dept Physiol & Neurobiol, Pazmany P Setany 1-C, H-1117 Budapest, Hungary (Reprint); Lorand Eotvos Univ, Dept Physiol & Neurobiol, H-1117 Budapest, Hungary; Hungarian Acad Sci, Biol Res Ctr, Inst Enzymol, H-1113 Budapest, Hungary
CYA Hungary
SO BRAIN RESEARCH, (22 OCT 2004) Vol. 1024, No. 1-2, pp. 150-158.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0006-8993.
DT Article; Journal
LA English
REC Reference Count: 59
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
STP KeyWords Plus (R): RAT HIPPOCAMPAL SLICES; MU-CALPAIN; SYNAPTIC PLASTICITY; CEREBRAL-ISCHEMIA; **GLUTAMATE** RECEPTORS; **FODRIN** PROTEOLYSIS; CRYSTAL-STRUCTURE; SILENT SYNAPSES; NMDA RECEPTORS; BRAIN-INJURY

L3 ANSWER 3 OF 19 MEDLINE on STN DUPLICATE 2
AN 2003464178 MEDLINE
DN PubMed ID: 12932846
TI Calpain induces proteolysis of neuronal cytoskeleton in ischemic gerbil forebrain.
AU Yokota Masayuki; Saido Takaomi C; Kamitani Hideki; Tabuchi Sadaharu; Satokata Ichiro; Watanabe Takashi
CS Department of Neurosurgery, School of Medicine, Tottori University, Tottori, Japan.. yokotans@grape.med.tottori-u.ac.jp
SO Brain research, (2003 Sep 12) 984 (1-2) 122-32.
Journal code: 0045503. ISSN: 0006-8993.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200312
ED Entered STN: 20031008
Last Updated on STN: 20031218
Entered Medline: 20031210
AB We investigated the relationship between the activity of calcium-dependent protease (calpain) and the ischemic neuronal damage. We also investigated the mechanism of ischemic resistance in astrocytes. In gerbil, a 10-min forebrain ischemia was induced by occlusion of both common carotid arteries. The calpain-induced proteolysis of cytoskeleton (**fodrin**) was examined by immunohistochemistry. Immunolocalization of micro and m-calpain was also examined. Intact **fodrin** was observed both in neurons and astrocytes, but proteolyzed **fodrin** was not observed in normal brain. Fifteen minutes after ischemia, proteolysis of **fodrin** took place in putamen, parietal cortex and hippocampal CA1. The proteolysis extended to thalamus 4 h after ischemia after which the immunoreactivity faded down in all areas except hippocampus. On day 7, the proteolysis was still observed only in hippocampus. Neurons with the proteolysis of soma resulted in neuronal death. Throughout the experiment, the proteolysis was not observed in astrocytes. micro -Calpain was observed only in neurons but m-calpain was observed both in neurons and astrocytes. The ischemia induced only micro -calpain activation, which resulted in **fodrin** proteolysis of neurons with differential spatial distribution and temporal course. The proteolysis was developed rapidly and was completed within 24 h in all vulnerable regions except hippocampal CA1. The proteolysis preceded the neuronal death. The mechanism of the proteolysis seemed to be involved by Ca(2+) influx via **glutamate** receptor and rapid neuronal death seemed reasonable. The reason why neuronal death in CA1 evolved slowly was not clarified. In astrocytes, **fodrin** was not proteolyzed by m-calpain. The low Ca(2+)-sensitivity of m-calpain may be the reason of ischemic resistance in astrocytes.

L3 ANSWER 4 OF 19 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2002:593614 HCAPLUS
DN 137:277123
TI Selective release of calpain produced αII-spectrin (α-fodrin)

breakdown products by acute neuronal cell death
AU Dutta, Satavisha; Chiu, Yuk Chun; Probert, Albert W.; Wang, Kevin K. W.
CS Laboratory of Neurobiochemistry, Department of Neuroscience Therapeutics, Pfizer Global Research and Development, Ann Arbor, MI, 48105, USA
SO Biological Chemistry (2002), 383(5), 785-791
CODEN: BICHF3; ISSN: 1431-6730
PB Walter de Gruyter GmbH & Co. KG
DT Journal
LA English
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT **Glutamate** receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(NMDA-binding; selective release of calpain produced αII-spectrin (α- **fodrin**) breakdown products by NMDA and kainate-induced rat cerebellar granular neuronal cell death)
IT **Glutamate** receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(kainate-binding; selective release of calpain produced αII-spectrin (α- **fodrin**) breakdown products by NMDA and kainate-induced rat cerebellar granular neuronal cell death)

L3 ANSWER 5 OF 19 MEDLINE on STN DUPLICATE 3
AN 2002706855 MEDLINE
DN PubMed ID: 12467876
TI Proteases involved in long-term potentiation.
AU Tomimatsu Yoshiro; Idemoto Satoru; Moriguchi Shigeki; Watanabe Shigenori; Nakanishi Hiroshi
CS Laboratory of Oral Aging Science, Division of Oral Biological Sciences, Faculty of Dental Sciences, Kyushu University, 812-8582, Fukuoka, Japan.
SO Life sciences, (2002 Dec 20) 72 (4-5) 355-61. Ref: 23
Journal code: 0375521. ISSN: 0024-3205.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200301
ED Entered STN: 20021217
Last Updated on STN: 20030128
Entered Medline: 20030127
AB Much attention has been paid to proteases involved in long-term potentiation (LTP). Calpains, Ca-dependent cysteine proteases, have first been demonstrated to be the mediator of LTP by the proteolytic cleavage of **fodrin**, which allows **glutamate** receptors located deep in the postsynaptic membrane to move to the surface. It is now generally considered that calpain activation is necessary for LTP formation in the cleavage of substrates such as protein kinase Czeta, NMDA receptors, and the **glutamate** receptor-interacting protein. Recent studies have shown that serine proteases such as tissue-type plasminogen activator (tPA), thrombin, and neuropsin are involved in LTP. tPA contributes to LTP by both receptor-mediated activation of cAMP-dependent protein kinase and the cleavage of NMDA receptors. Thrombin induces a proteolytic activation of PAR-1, resulting in activation of protein kinase C, which reduces the voltage-dependent Mg2+ blockade of NMDA receptor-channels. On the other hand, neuropsin may act as a regulatory molecule in LTP via its proteolytic degradation of extracellular matrix protein such as fibronectin. In addition to such neuronal proteases, proteases secreted from microglia such as tPA may also contribute to LTP. The enzymatic activity of each protease is strictly regulated by endogenous inhibitors and other factors in the brain. Once activated, proteases can irreversibly cleave peptide bonds. After cleavage, some substrates are inactivated and others are activated to gain new functions. Therefore, the issue to identify substrates for each protease is very important to understand the molecular basis of LTP.

L3 ANSWER 6 OF 19 MEDLINE on STN DUPLICATE 4
AN 2001636608 MEDLINE

DN PubMed ID: 11509555
TI Synaptic scaffolding proteins in rat brain. Ankyrin repeats of the multidomain Shank protein family interact with the cytoskeletal protein alpha-fodrin.
AU Bockers T M; Mameza M G; Kreutz M R; Bockmann J; Weise C; Buck F; Richter D; Gundelfinger E D; Kreienkamp H J
CS Arbeitsgruppe Molekulare Neurobiologie, Institut fur Anatomie, Westfalische Wilhelms-Universitat, 48149 Munster, Germany,.
SO Journal of biological chemistry, (2001 Oct 26) 276 (43) 40104-12. Electronic Publication: 2001-08-16. Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200112
ED Entered STN: 20011107 Last Updated on STN: 20030105 Entered Medline: 20011207
AB The postsynaptic density is the ultrastructural entity containing the neurotransmitter reception apparatus of excitatory synapses in the brain. A recently identified family of multidomain proteins termed Src homology 3 domain and ankyrin repeat-containing (Shank), also known as proline-rich synapse-associated protein/somatostatin receptor-interacting protein, plays a central role in organizing the subsynaptic scaffold by interacting with several synaptic proteins including the **glutamate** receptors. We used the N-terminal ankyrin repeats of Shank1 and -3 to search for interacting proteins by yeast two-hybrid screening and by affinity chromatography. By cDNA sequencing and mass spectrometry the cytoskeletal protein alpha-**fodrin** was identified as an interacting molecule. The interaction was verified by pull-down assays and by coimmunoprecipitation experiments from transfected cells and brain extracts. Mapping of the interacting domains of alpha-**fodrin** revealed that the highly conserved spectrin repeat 21 is sufficient to bind to the ankyrin repeats. Both interacting partners are coexpressed widely in the rat brain and are colocalized in synapses of hippocampal cultures. Our data indicate that the Shank1 and -3 family members provide multiple independent connections between synaptic **glutamate** receptor complexes and the cytoskeleton.

L3 ANSWER 7 OF 19 MEDLINE on STN DUPLICATE 5
AN 2001158843 MEDLINE
DN PubMed ID: 11181835
TI IPF, a vesicular uptake inhibitory protein factor, can reduce the Ca(2+)-dependent, evoked release of glutamate, GABA and serotonin.
AU Tamura Y; Ozkan E D; Bole D G; Ueda T
CS Mental Health Research Institute, The University of Michigan, Ann Arbor, Michigan, USA.
NC MH 15794-18 (NIMH) NS 26884 (NINDS) NS 36656 (NINDS)
SO Journal of neurochemistry, (2001 Feb) 76 (4) 1153-64. Journal code: 2985190R. ISSN: 0022-3042.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200103
ED Entered STN: 20010404 Last Updated on STN: 20010404 Entered Medline: 20010322
AB Synaptic vesicles in the nerve terminal play a pivotal role in neurotransmission. Neurotransmitter accumulation into synaptic vesicles is catalyzed by distinct vesicular transporters, harnessing an electrochemical proton gradient generated by V-type proton-pump ATPase. However, little is known about regulation of the transmitter pool size, particularly in regard to amino acid neurotransmitters. We previously provided evidence for the existence of a potent endogenous inhibitory protein factor (IPF), which causes reduction of **glutamate** and

GABA accumulation into isolated, purified synaptic vesicles. In this study we demonstrate that IPF is concentrated most in the synaptosomal cytosol fraction and that, when introduced into the synaptosome, it leads to a decrease in calcium-dependent exocytotic (but not calcium-independent) release of **glutamate** in a concentration-dependent manner. In contrast, alpha-**fodrin** (non-erythroid spectrin), which is structurally related to IPF and thought to serve as the precursor for IPF, is devoid of such inhibitory activity. Intrasynaptosomal IPF also caused reduction in exocytotic release of GABA and the monoamine neurotransmitter serotonin. Whether IPF affects vesicular storage of multiple neurotransmitters in vivo would depend upon the localization of IPF. These results raise the possibility that IPF may modulate synaptic transmission by acting as a quantal size regulator of one or more neurotransmitters.

L3 ANSWER 8 OF 19 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:699214 HCAPLUS
DN 133:286460
TI Fodrin compositions and methods for the inhibition of neurotransmitter uptake by synaptic vesicles
IN Ueda, Tetsufumi; Ozkan, Eric D.
PA Regents of the University of Michigan, USA
SO U.S., 37 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6127520	A	20001003	US 1997-840006	19970415

PRAI US 1997-840006 19970415
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB **Fodrin** compns. and methods for treating neurosynaptic disorder in a subject are described. More specifically, compns. and methods for inhibiting **glutamate** uptake by synaptic vesicles in a subject are set forth. In one embodiment, the composition is inhibitory protein factor (IPF) and the subject is a human.
ST **fodrin glutamate** uptake synaptic vesicles
IT Biological transport
(uptake, of **glutamate**; **fodrin** compns. and methods for the inhibition of neurotransmitter uptake by synaptic vesicles)

L3 ANSWER 9 OF 19 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 6
AN 1999:184092 SCISEARCH
GA The Genuine Article (R) Number: 170VD
TI Species differences in fodrin proteolysis in the ischemic brain
AU Kitagawa K (Reprint); Matsumoto M; Saido T C; Ohtsuki T; Kuwabara K; Yagita Y; Mabuchi T; Yanagihara T; Hori M
CS OSAKA UNIV, SCH MED, DEPT INTERNAL MED 1, DIV STROKOL, 2-2 YAMADAOKA, SUITA, OSAKA 565, JAPAN (Reprint); OSAKA UNIV, SCH MED, DEPT NEUROL, OSAKA, JAPAN; TOKYO METROPOLITAN INST MED SCI, DEPT BIOL MOL, TOKYO 113, JAPAN
CYA JAPAN
SO JOURNAL OF NEUROSCIENCE RESEARCH, (1 MAR 1999) Vol. 55, No. 5, pp. 643-649.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0360-4012.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 44
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
ST Author Keywords: calpain; **fodrin**; microtubule-associated protein 2; **glutamate**; ischemia

L3 ANSWER 10 OF 19 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on

STN DUPLICATE 7
AN 2000:8266 SCISEARCH
GA The Genuine Article (R) Number: 267JJ
TI Differential changes in glutamatergic transmission via N-methyl-D-aspartate receptors in the hippocampus and striatum of rats behaviourally sensitized to methamphetamine
AU Yamamoto H; Kitamura N; Lin X H; Ikeuchi Y; Hashimoto T; Shirakawa O (Reprint); Maeda K
CS KOBE UNIV, SCH MED, DEPT PSYCHIAT & NEUROL, CHUO KU, 7-5-1 KUSUNOKI CHO, KOBE, HYOGO 6500017, JAPAN (Reprint); KOBE UNIV, SCH MED, DEPT PSYCHIAT & NEUROL, CHUO KU, KOBE, HYOGO 6500017, JAPAN; SHINKO HOSP, DEPT PSYCHIAT, KOBE, HYOGO 6510072, JAPAN
CYA JAPAN
SO INTERNATIONAL JOURNAL OF NEUROPSYCHOPHARMACOLOGY, (SEP 1999) Vol. 2, No. 3, pp. 155-163.
Publisher: CAMBRIDGE UNIV PRESS, 40 WEST 20TH STREET, NEW YORK, NY 10011-4211.
ISSN: 1461-1457.
DT Article; Journal
LA English
REC Reference Count: 48
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We searched for changes in glutamatergic transmission via N-methyl-D-aspartate (NMDA) receptors in the hippocampus and striatum of rats behaviourally sensitized to methamphetamine (Meth). Prior to being given a challenge dose of Meth (2 mg/kg, s.c.), the rats were given Meth (4 mg/kg, s.c.) five times a week for 3 wk. Seven days after the challenge test, we examined **glutamate** (Glu) release from hippocampal and striatal slices evoked by 30 mM KCl, and NMDA-evoked dopamine (DA) release from striatal slices. We further immunoquantified NMDAR1, R2A and R2B receptors as well as the **fodrin** alpha-subunit, a 240 kDa cytoskeletal protein that is cleaved to form 150 kDa limited proteolytic fragments by NMDA receptor stimulation. In the study of KCl-evoked Glu release, Glu release from the hippocampus was 31% lower in the Meth-sensitized rats than in the control rats, while Glu release from the striatum was 34% higher in the Meth-sensitized rats. NMDAR1, R2A and R2B immunoreactivities in the striatum were significantly lower in the Meth-sensitized rats (by 12, 13 and 12%, respectively) than those in the control rats. However, no differences in the immunoreactivities were found for the hippocampus. Immunoquantification of the **fodrin** a-subunit in the hippocampus revealed that 150 kDa fragments were significantly lower (by 10%) in the Meth-sensitized rats than in the control rats. In contrast to the control rats, NMDA-evoked DA release from the striatum was diminished in the Meth-sensitized rats. These results indicate that the activity of the Glu system is functionally decreased in the hippocampus of Meth-sensitized rats, whereas the Glu system in the striatum of Meth-sensitized mts shows adaptive and functional changes in the receptors in response to the increased Glu release.

L3 ANSWER 11 OF 19 MEDLINE on STN DUPLICATE 8
AN 1999021714 MEDLINE
DN PubMed ID: 9804614
TI 1-Methyl-4-phenylpyridinium induces autocrine excitotoxicity, protease activation, and neuronal apoptosis.
AU Leist M; Volbracht C; Fava E; Nicotera P
CS Faculty of Biology, Chair of Molecular Toxicology, University of Konstanz, D-78457 Konstanz, Germany.
SO Molecular pharmacology, (1998 Nov) 54 (5) 789-801.
Journal code: 0035623. ISSN: 0026-895X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199812
ED Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981216
AB The pathogenesis of several neurodegenerative diseases may involve indirect excitotoxic mechanisms, where **glutamate** receptor

overstimulation is a secondary consequence of initial functional defects of neurons (e.g., impairment of mitochondrial energy generation). The neurotoxin 1-methyl-4-phenylpyridinium (MPP+) and other mitochondrial inhibitors (e.g., rotenone or 3-nitropropionic acid) elicited apoptosis in cerebellar granule cell cultures via stimulation of autocrine excitotoxicity. Cell death, increase in intracellular Ca2+ concentration, release of cytochrome c, and all biochemical and morphological signs of apoptosis were prevented by blockade of the N-methyl-D-aspartate receptor with noncompetitive, glycine-site or **glutamate**-site inhibitors. In addition, MPP+-induced apoptosis was reduced by high Mg2+ concentrations in the medium or by inhibiting exocytosis with clostridial neurotoxins. Two classes of cysteine proteases were involved in the execution of cell death: caspases and calpains. Inhibitors of either class of proteases prevented cell death, cleavage of intracellular proteins (i.e., **fodrin**), and the appearance of typical features of apoptosis such as phosphatidylserine translocation or DNA fragmentation. However, protease inhibitors did not interfere with the initial intracellular Ca2+ concentration increase. We suggest that MPP+ as well as other mitochondrial inhibitors trigger indirect excitotoxic processes, which lead to Ca2+ overload, protease activation, and subsequent neuronal apoptosis.

L3 ANSWER 12 OF 19 MEDLINE on STN DUPLICATE 9
AN 97268710 MEDLINE
DN PubMed ID: 9108118
TI A protein factor that inhibits ATP-dependent glutamate and gamma-aminobutyric acid accumulation into synaptic vesicles: purification and initial characterization.
AU Ozkan E D; Lee F S; Ueda T
CS Mental Health Research Institute, University of Michigan, Ann Arbor 48109-0720, USA.
NC GM 07863 (NIGMS)
NS 26884 (NINDS)
SO Proceedings of the National Academy of Sciences of the United States of America, (1997 Apr 15) 94 (8) 4137-42.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199705
ED Entered STN: 19970602
Last Updated on STN: 19970602
Entered Medline: 19970522
AB **Glutamate**, the major excitatory neurotransmitter in the mammalian central nervous system, is transported into and stored in synaptic vesicles. We have purified to apparent homogeneity a protein from brain cytosol that inhibits **glutamate** and gamma-aminobutyric acid uptake into synaptic vesicles and have termed this protein "inhibitory protein factor" (IPF). IPF refers to three distinct proteins with relative molecular weights of 138,000 (IPF alpha), 135,000 (IPF beta), and 132,000 (IPF gamma), respectively. Gel filtration and sedimentation data suggest that all three proteins share an elongated structure, identical Stokes radius (60 A), and identical sedimentation coefficient (4.3 S). Using these values and a partial specific volume of 0.716 ml/g, we determined the native molecular weight for IPF alpha to be 103,000. Partial sequence analysis shows that IPF alpha is derived from alpha **fodrin**, a protein implicated in several diverse cellular activities. IPF alpha inhibits ATP-dependent **glutamate** uptake into purified synaptic vesicles with an IC50 of approximately 26 nM, while showing no ability to inhibit ATP-independent uptake at concentrations up to 100 nM. Moreover, IPF alpha inhibited neither norepinephrine uptake into chromaffin vesicles nor Na+-dependent **glutamate** uptake into synaptosomes. However, IPF alpha inhibited uptake of gamma-aminobutyric acid into synaptic vesicles derived from spinal cord, suggesting that inhibition may not be limited to glutamatergic systems. We propose that IPF could be a novel component of a presynaptic regulatory system. Such a system might modulate neurotransmitter accumulation into synaptic vesicles and thus regulate the overall efficacy of neurotransmission.

L3 ANSWER 13 OF 19 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 95:354228 SCISEARCH
GA The Genuine Article (R) Number: QX925
TI INDUCTION OF CALPAIN-MEDIATED SPECTRIN FRAGMENTS BY PATHOGENIC TREATMENTS IN LONG-TERM HIPPOCAMPAL SLICES
AU BAHR B A (Reprint); TIRIVEEDHI S; PARK G Y; LYNCH G
CS UNIV CALIF IRVINE, CTR NEUROBIOL LEARNING & MEMORY, IRVINE, CA, 92717 (Reprint)
CYA USA
SO JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, (MAY 1995) Vol. 273, No. 2, pp. 902-908.
ISSN: 0022-3565.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 34
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
STP KeyWords Plus (R): BRAIN SPECTRIN; ORGANOTYPIC CULTURES; **GLUTAMATE** RECEPTOR; PROTEOLYSIS; PROTEASE; **FODRIN**; SITE; POTENTIATION; TRIMETHYLTIN; DEGRADATION

L3 ANSWER 14 OF 19 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
AN 95:555925 SCISEARCH
GA The Genuine Article (R) Number: RP369
TI CALPAIN AS A NOVEL TARGET FOR TREATING ACUTE NEURODEGENERATIVE DISORDERS
AU BARTUS R T (Reprint); ELLIOTT P J; HAYWARD N J; DEAN R L; HARBESON S; STRAUB J A; LI Z; POWERS J C
CS ALKERMES INC, 64 SIDNEY ST, CAMBRIDGE, MA, 02139 (Reprint); GEORGIA INST TECHNOL, SCH CHEM, ATLANTA, GA, 30332
CYA USA
SO NEUROLOGICAL RESEARCH, (AUG 1995) Vol. 17, No. 4, pp. 249-258.
ISSN: 0161-6412.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 40
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
STP KeyWords Plus (R): **GLUTAMATE** NEUROTOXICITY; BRAIN SPECTRIN; PROTEOLYSIS; ISCHEMIA; INHIBITORS; DAMAGE; **FODRIN**; DEGRADATION; PROTEASES

L3 ANSWER 15 OF 19 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1991:624343 HCAPLUS
DN 115:224343
TI Excitatory amino acids induce calcium/calpain-I-dependent proteolysis of brain spectrin in cultured central neurons
AU Gallo, V.; Di Stasi, A. M. M.; Ceccarini, M.; Petrucci, T. C.
CS Lab. Physiopathol., Ist. Super. Sanita, Rome, 00161, Italy
SO Fidia Research Foundation Symposium Series (1991), 5(Excitatory Amino Acids), 267-73
CODEN: FRFSEL; ISSN: 1040-0451
DT Journal
LA English
AB The authors studied the expression of **fodrin** and its regulation during development in a homogeneous population of cultured cerebellar neurons, the granule cells. A large number of studies have demonstrated that cerebellar granule cells in culture express excitatory amino acid receptors and channels and are susceptible to the toxic action of **glutamate.** The authors also investigated, if **glutamate** receptor activation and subsequent opening of channels permeable to Ca2+ ions would cause calpain I-induced degradation of **fodrin** in cerebellar granule cells. Finally, it was examined if the proteolytic cleavage of **fodrin** is directly responsible for the cell death observed after exposure to excitatory amino acids.

L3 ANSWER 16 OF 19 HCAPLUS COPYRIGHT 2005 ACS on STN

AN 1990:212559 HCAPLUS
DN 112:212559
TI Axon and axolemma
AU Matsumoto, Gen; Ichikawa, Michinori; Tsukita, Shoichiro; Arai, Takao
CS Mol. Cell. Neurosci. Sect., Electrotech. Lab., Tsukuba, 305, Japan
SO Tanpakushitsu Kakusan Koso (1990), 35(4), 454-63
CODEN: TAKKAJ; ISSN: 0039-9450
DT Journal; General Review
LA Japanese
AB A review with 40% on the structure and function of the axolemma. The axon is different from the dendrite in its subcellular structures including cytoskeletons and organella as well as its function and growing mechanism. Mol. structures of the subunits of the Na+ channel and their relation to physiol. activities of the channel such as voltage sensor, Na+ permeation and cycles of activation and inactivatin are under investigation. Na+ channel is localized in nodes of Ranvier and K+ channel in myelin region. The former is supposed to be connected to cytoskeletal proteins such as **fodrin.** Astrocytes, whose processes attach to the nodes of Ranier and the high electron d. region of nonmyelinated axons, also have ion channels, nerve transmitter receptors and their second messengers and non-NMDA type **glutamate** receptors. The actin filament region of the squid giant axon membrane is rich in the Na+ channel and bound to Schwann cell which has non-NMDA **glutamate** receptors and regulates K+ concentration outside axons.

L3 ANSWER 17 OF 19 MEDLINE on STN DUPLICATE 10
AN 85111110 MEDLINE
DN PubMed ID: 2982099
TI Regulation of **glutamate** receptor binding by the cytoskeletal protein **fodrin.**
AU Siman R; Baudry M; Lynch G
NC MH 190793-12 (NIMH)
SO Nature, (1985 Jan 17-23) 313 (5999) 225-8.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198502
ED Entered STN: 19900320
Last Updated on STN: 20000303
Entered Medline: 19850225
TI Regulation of **glutamate** receptor binding by the cytoskeletal protein **fodrin.**
AB The erythrocyte cytoskeleton, which consists primarily of a meshwork of spectrin and actin, controls cell shape and the disposition of proteins within the membrane. Proteins similar to spectrin have recently been found in diverse cells and tissues, and it is possible that they mediate the capping of cell-surface receptors, although this has not been demonstrated directly. In neurones, the spectrin-like protein **fodrin** lines the cortical cytoplasm and may link actin filaments to the membrane. **Fodrin** has been hypothesized to regulate the number of receptor binding sites on neuronal membranes for the putative neurotransmitter L-**glutamate.** Micromolar calcium concentrations activate the thiol protease calpain I, induce **fodrin** degradation and more than double the density of **glutamate** binding sites; these effects are all blocked by thiol protease inhibitors. We have now used specific antibodies to examine further the role of **fodrin** proteolysis in regulating **glutamate** receptors. We report that **fodrin** antibodies block the **fodrin** degradation and increase in **glutamate** binding normally induced by calcium, and so provide direct evidence for control of membrane receptors by a non-erythroid spectrin.

L3 ANSWER 18 OF 19 MEDLINE on STN DUPLICATE 11
AN 84196409 MEDLINE
DN PubMed ID: 6144182
TI The biochemistry of memory: a new and specific hypothesis.
AU Lynch G; Baudry M

NC AG 00538 (NIA)
MH 19793-12 (NIMH)
NH 00358-03
SO Science, (1984 Jun 8) 224 (4653) 1057-63.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198406
ED Entered STN: 19900319
Last Updated on STN: 20000303
Entered Medline: 19840621
AB Recent studies have uncovered a synaptic process with properties required for an intermediate step in memory storage. Calcium rapidly and irreversibly increases the number of receptors for **glutamate** (a probable neurotransmitter) in forebrain synaptic membranes by activating a proteinase (calpain) that degrades **fodrin**, a spectrin-like protein. This process provides a means through which physiological activity could produce long-lasting changes in synaptic chemistry and ultrastructure. Since the process is only poorly represented in the brain stem, it is hypothesized to be responsible for those forms of memory localized in the telencephalon.

L3 ANSWER 19 OF 19 MEDLINE on STN DUPLICATE 12
AN 83262176 MEDLINE
DN PubMed ID: 6307724
TI Regulation by calcium ions of glutamate receptor binding in hippocampal slices.
AU Baudry M; Siman R; Smith E K; Lynch G
SO European journal of pharmacology, (1983 Jun 3) 90 (2-3) 161-8.
Journal code: 1254354. ISSN: 0014-2999.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198309
ED Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19830923
AB Hippocampal slices were incubated in a Krebs-bicarbonate buffer with various concentrations of calcium and [3H]**glutamate** receptor binding was measured in crude synaptic membranes derived from these slices. Increasing the calcium concentration from 0 to 2.5 mM resulted in a 2.2-fold increase in the maximal number of the Na-independent [3H] **glutamate** binding sites without changes in their affinity for [3H] **glutamate.** This effect was totally blocked by the addition of the protease inhibitor leupeptin (50 microM) to the slice incubation medium. No effect was observed on the Na-dependent [3H]**glutamate** binding nor on the Na-independent [3H]**glutamate** binding measured in the presence of a concentration of calcium of 250 microM. Increasing the calcium concentration also resulted in an increased proteolytic activity which was inhibited by about 70% by the addition of leupeptin. Finally, increasing the calcium concentration induced the degradation of high-molecular weight proteins, the microtubule-associated proteins (MAPs) and the 220 000 dalton doublet protein corresponding to **fodrin.** Both effects were partially prevented by the addition of leupeptin in the slice incubation medium. These results indicate that the same calcium-dependent processes which were previously shown to regulate [3H] **glutamate** receptor binding to hippocampal membranes occur in the hippocampal slice preparation, and they suggest a mechanism by which fluctuations in calcium levels can activate a calcium-dependent proteinase, the degradation of cytoskeletal-associated proteins and the unmasking of additional **glutamate** receptors. The participation of such processes in various forms of plasticity is discussed.

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=> s fodrin(P)synaptic
L4 79 FODRIN(P) SYNAPTIC

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PROCESSING COMPLETED FOR L4
L5 36 DUP REM L4 (43 DUPLICATES REMOVED)

=> d bib hit 20-
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L5 ANSWER 20 OF 36 MEDLINE on STN DUPLICATE 9
AN 90186458 MEDLINE
DN PubMed ID: 2312414
TI Nonerythroid spectrin (fodrin) is a prominent component of the cochlear hair cells.
AU Ylikoski J; Pirvola U; Narvanen O; Virtanen I
CS Department of Anatomy, University of Helsinki, Finland.
SO Hearing research, (1990 Jan) 43 (2-3) 199-203.
Journal code: 7900445. ISSN: 0378-5955.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199004
ED Entered STN: 19900601
Last Updated on STN: 19900601
Entered Medline: 19900413
AB We studied the distribution of nonerythroid spectrin, **fodrin**, in surface preparations and cryosections of the cochlear hair cells as well as isolated hair cells of the guinea pig by using a monoclonal antibody (Mab) reacting with Mr 240 kD alpha-**fodrin** polypeptide. The Mab gave a strong reaction with the cuticular plate of both the inner and

outer hair cells (IHCs and OHCs). Stereocilia were nonreactive and only a weak cell surface reaction was seen in the supporting cells. In the outer hair cells the upper turns of the cochlea, **fodrin** was observed in a cytoplasmic spiralling structure extending from the cuticular plate towards the cell nucleus. Some labelling was also seen along the cell surface membrane and in the **synaptic** region. The results suggest that **fodrin** may be an important constituent in the active processes of hair cells such as cell motility and/or signal transduction.

L5 ANSWER 21 OF 36 HCAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 10
AN 1989:90948 HCAPLUS
DN 110:90948
TI The cytoskeletal architecture of the presynaptic terminal and molecular structure of synapsin 1
AU Hirokawa, Nobutaka; Sobue, Kenji; Kanda, Keiko; Harada, Akihiro; Yorifuji, Hiroshi
CS Sch. Med., Univ. Tokyo, Tokyo, Japan
SO Journal of Cell Biology (1989), 108(1), 111-26
CODEN: JCLBA3; ISSN: 0021-9525
DT Journal
LA English

AB The cytoskeletal architecture and its relationship with **synaptic** vesicles in synapses was examined by quick-freeze deep-etch electron microscopy (QF·DE). The main cytoskeletal elements in the presynaptic terminals (neuromuscular junction, elec. organ, and cerebellar cortex) were actin filaments and microtubules. The actin filaments formed a network and frequently were associated closely with the presynaptic plasma membranes and active zones. Short, linking strands .apprx.30 nm long were found between actin and **synaptic** vesicles, between microtubules and **synaptic** vesicles. Fine strands (30-60 nm) were also found between **synaptic** vesicles. Frequently spherical structures existed in the middle of the strands between **synaptic** vesicles. Another kind of strand (.apprx.100 nm long, thinner than the actin filaments) between **synaptic** vesicles and plasma membranes was also observed The mol. structure of synapsin 1 and its relationship with actin filaments, microtubules, and **synaptic** vesicles was examined in vitro using the low-angle rotary-shadowing technique and QF·DE. The synapsin 1 mol., .apprx.47 nm long, was composed of a head (.apprx.14 nm diameter) and a tail (.apprx.33 nm long), having a tadpolelike appearance. The high resolution provided by QF·DE revealed that a single synapsin 1 crosslinked actin filaments and linked actin filaments with **synaptic** vesicles, forming .apprx.30-nm short strands. The head was on the actin and the tail was attached to the **synaptic** vesicle or actin filament. Microtubules were also crosslinked by a single synapsin 1, which also connected a microtubule to **synaptic** vesicles, forming .apprx.30 nm strands. The spherical head was on the microtubule and the tail was attached to the **synaptic** vesicles or to microtubules. **Synaptic** vesicles incubated with synapsin 1 were linked with each other via fine short fibrils and spherical structures from which 2 or 3 fibrils radiated and crosslinked **synaptic** vesicles were frequently identified. The localization of synapsin 1 was examined using ultracryomicrotomy and colloidal Au-immunocytochem. of anti-synapsin 1 IgG. Synapsin 1 was exclusively localized in the regions occupied by **synaptic** vesicles. Statistical analyses indicated that synapsin 1 is located mostly at least .apprx.30 nm away from the presynaptic membrane. These data derived via 3 different approaches suggest that synapsin 1 could be a main element of short linkages between actin filaments and **synaptic** vesicles, between microtubules and **synaptic** vesicles, and between **synaptic** vesicles in the nerve terminals. The longer strands (.apprx.100 nm) associated with presynaptic membrane could consist of other proteins, most probably **fodrin**, judging from its structure. Because phosphorylation of synapsin 1 by Ca2+/calmodulin-dependent kinase detaches synapsin 1 from vesicles it could release **synaptic** vesicles from actin filaments, microtubules, and other **synaptic** vesicles, and thus increase the mobility of **synaptic** vesicles to the presynaptic membrane after depolarization-dependent influx of Ca2+ into the presynaptic terminal.

L5 ANSWER 22 OF 36 MEDLINE on STN
AN 89004128 MEDLINE
DN PubMed ID: 3048888
TI Spectrin and related molecules.
AU Goodman S R; Krebs K E; Whitfield C F; Riederer B M; Zagon I S
CS Cell and Molecular Biology Center, Milton S. Hershey Medical Center, Pennsylvania State University.
SO CRC critical reviews in biochemistry, (1988) 23 (2) 171-234. Ref: 389
Journal code: 0330403. ISSN: 0045-6411.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 198811
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19881123
AB This review begins with a complete discussion of the erythrocyte spectrin membrane skeleton. Particular attention is given to our current knowledge of the structure of the RBC spectrin molecule, its synthesis, assembly, and turnover, and its interactions with spectrin-binding proteins (ankyrin, protein 4.1, and actin). We then give a historical account of the discovery of nonerythroid spectrin. Since the chicken intestinal form of spectrin (TW260/240) and the brain form of spectrin (**fodrin**) are the best characterized of the nonerythroid spectrins, we compare these molecules to RBC spectrin. Studies establishing the existence of two brain spectrin isoforms are discussed, including a description of the location of these spectrin isoforms at the light- and electron-microscope level of resolution; a comparison of their structure and interactions with spectrin-binding proteins (ankyrin, actin, synapsin I, amelin, and calmodulin); a description of their expression during brain development; and hypotheses concerning their potential roles in axonal transport and **synaptic** transmission.

L5 ANSWER 23 OF 36 MEDLINE on STN DUPLICATE 11
AN 88061579 MEDLINE
DN PubMed ID: 3119792
TI Axonal transport of synapsin I-like proteins in rabbit retinal ganglion cells.
AU Baitinger C; Willard M
CS Department of Anatomy and Neurobiology, Washington University School of Medicine, Saint Louis, Missouri 63110.
NC EYO 2682 (NEI)
SO Journal of neuroscience : official journal of the Society for Neuroscience, (1987 Nov) 7 (11) 3723-35.
Journal code: 8102140. ISSN: 0270-6474.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198801
ED Entered STN: 19900305
Last Updated on STN: 19900305
Entered Medline: 19880119
AB Synapsin I is a neuronal phosphoprotein that is associated with the cytoplasmic surface of small, clear **synaptic** vesicles in neuronal **synaptic** terminals; it may play an important role in **synaptic** transmission. In vitro, it can interact with **fodrin**, a relative of the erythrocyte protein spectrin. We have investigated the delivery of synapsin I from its site of synthesis in neuronal cell bodies to **synaptic** terminals by means of the process of axonal transport. We labeled the newly synthesized proteins of rabbit retinal ganglion cells by injecting 35S-methionine into the vitreous humour, and subsequently observed the appearance of radioactive synapsin I (identified by its 2-dimensional electrophoretic mobility) in tissues containing the axons and **synaptic** terminals of these neurons. A portion of the newly synthesized synapsin I was axonally

transported at the velocity of the most rapidly transported (group I) proteins, which comprise membrane-associated proteins and may include elements of **synaptic** vesicles. However, the subsequent time course of labeling of synapsin I in the axons suggests that greater than 90% of the axonally transported synapsin I may comprise 2 additional populations--one transported rapidly, the other slowly--that are released from the cell bodies only after a delay of more than 1 d. The delayed, slowly transported population moves at the velocity (approximately 6 mm/d) of groups III and IV (which include **fodrin** and other proteins of the membrane cytoskeleton). We consider whether such distinct populations may correspond to functionally specialized variants of synapsin I-like proteins that may be transported in association with different organelles. The electrophoretic mobility of labeled synapsin I-like proteins in the axons changed subtly with time. Additional subtle differences between labeled synapsin I-like proteins in the axons and the terminal-containing tissues suggest that certain posttranslational modifications occur specifically in the terminals.

L5 ANSWER 24 OF 36 MEDLINE on STN DUPLICATE 12
AN 87300882 MEDLINE
DN PubMed ID: 3621001
TI Translocations of fodrin and its binding proteins.
AU Willard M; Baitinger C; Cheney R
NC EY02682 (NEI)
SO Brain research bulletin, (1987 Jun) 18 (6) 817-24.
Journal code: 7605818. ISSN: 0361-9230.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198710
ED Entered STN: 19900305
Last Updated on STN: 19970203
Entered Medline: 19871016
AB **Fodrin**, a protein related to erythrocyte spectrin, redistributes within the cell in certain situations. We compare such movements of **fodrin** and several **fodrin** binding proteins during the processes of axonal transport in neurons, and capping of surface proteins in lymphocytes. In neurons, three different populations of newly synthesized **fodrin** appear to be transported down the axons at different velocities corresponding to those of groups of transported proteins designated II, IV, and V. Actin, which can interact with **fodrin**, is transported at the velocity of group IV. Synapsin, a component of **synaptic** vesicles, is also reported to bind to **fodrin.** One population of synapsin is transported more rapidly than **fodrin**, at the velocity of group I: two additional populations of transported synapsin may overlap **fodrin** in groups II and IV. We consider possible functional associations of these different populations of **fodrin** and **fodrin** binding proteins. We note that the transport of group IV proteins resembles in certain respects the process of capping in lymphocytes, suggesting the possibility of a common mechanism. We outline one of several possible mechanisms.

L5 ANSWER 25 OF 36 MEDLINE on STN DUPLICATE 13
AN 88089741 MEDLINE
DN PubMed ID: 3694236
TI Postnatal development of immunohistochemically localized spectrin-like protein (calspectin or fodrin) in the rat visual cortex: its excessive expression in developing cortical neurons.
AU Kimura F; Tsumoto T; Sobue K
CS Department of Neurophysiology, Osaka University Medical School, Japan.
SO Journal of neurocytology, (1987 Oct) 16 (5) 649-65.
Journal code: 0364620. ISSN: 0300-4864.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198802

ED Entered STN: 19900305
Last Updated on STN: 19900305
Entered Medline: 19880225

AB Postnatal development of the expression and localization of a membrane-associated cytoskeletal protein, calspectin (**fodrin** or brain spectrin), in the visual cortex, was immunohistochemically studied in newborn to adult rats, by using an anti-calspectin antibody. At birth, calspectin-immunoreactivity was already present at the plasma membrane and in the cytoplasm of neurons which were mostly pyramidal cells located in the upper part of the cortical subplate. Immature neurons located in the cortical plate were not stained by the antibody, suggesting that calspectin is expressed only in neurons which have differentiated or are differentiating. At postnatal days 2 to 7, immunoreactive neurons were dramatically increased in layers V and VI and very intense labelling was seen in the apical dendrites of layer V pyramidal cells. Most of the stained processes of these and other neurons showed signs of rapid dendritic growth, i.e. non-terminal as well as terminal growth cones and filopodia. At days 10 to 17, dendrites of pyramidal cells in layers II and III became clearly detectable, although still slender. At days 24 to 34, the basal dendrites of pyramidal cells in layers II, III and V became intensely immunoreactive and dendritic spines were visualized by the antibody. In the adult, however, the calspectin immunoreactivity became very weak and spines were not recognizable. At all the ages, axons and neuroglia were unstained. Also, most of the neurons in layer IV of the cortex were not immunoreactive. These results suggest that calspectin is most abundantly expressed in growing parts of the dendrites and spines. A hypothesis that calspectin may play a role in **synaptic** plasticity in the developing visual cortex is discussed.

L5 ANSWER 26 OF 36 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1987:174021 HCAPLUS
DN 106:174021
TI Effects of acetylcholinesterase inhibition on cholinergic transmission in the hippocampal slice
AU Lynch, Gary
CS Cent. Neurobiol. Learn. Mem., Univ. California, Irvine, CA, USA
SO Report (1986), AFOSR-TR-86-0299; Order No. AD-A169047/8/GAR, 25 pp.
Avail.: NTIS
From: Gov. Rep. Announce. Index (U. S.) 1986, 86(21), Abstr. No. 646,534
DT Report
LA English

AB Mechanism used by brain cells to change their functional interconnections and their possible involvement in neuropathol. were studied in hippocampal slices. Prolonged exposure to acidic amino acid transmitters caused functional desensitization of extrasynaptic receptors and inhibition of the second messenger system. It was also found that calpain degradation of the brain structural protein spectrin irreversibly changes amino acid receptors and that calpain is concentrated in **synaptic** regions of the brain. Spectrin was rapidly synthesized, inserted into membrane domains and apparently digested by calpain. Calmodulin accelerated the calpain-**fodrin** interactions in tissues studies.

L5 ANSWER 27 OF 36 MEDLINE on STN DUPLICATE 14
AN 85133856 MEDLINE
DN PubMed ID: 2579219
TI A regional analysis of alpha-spectrin in the isolated Mauthner neuron and in isolated axons of the goldfish and rabbit.
AU Koenig E; Repasky E
SO Journal of neuroscience : official journal of the Society for Neuroscience, (1985 Mar) 5 (3) 705-14.
Journal code: 8102140. ISSN: 0270-6474.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198504
ED Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850412

AB Isolated dendrites, somata, and desheathed axons of the goldfish Mauthner neuron (M-cell), in addition to other isolated myelin sheath-free axons of the goldfish spinal cord and of rabbit lumbar ventral roots, were shown by immunochemical and immunofluorescence techniques to contain alpha-spectrin (**fodrin**). alpha-Spectrin appeared to be organized as a randomly distributed reticular network, localized to the surface of isolated neuronal cellular structures. In addition, alpha-spectrin was also distributed nonrandomly at specialized cellular sites. These sites included **synaptic** junctions and morphologically differentiated nodes of Ranvier (i.e., rabbit axons, but not goldfish axons). At the latter sites, it is possible to demonstrate that alpha-spectrin is co-localized with F-actin, as indicated by a striking correspondence of fluorescent images due to double labeling, using the indirect immunofluorescence technique with alpha-spectrin antiserum, and direct binding of F-actin by rhodamine-conjugated palloidin. However, the spectrin-actin network at **synaptic** junctions appears to be distributed over the entire area of junctional contact and is not just restricted to postsynaptic densities. The possibility of a duality of roles of spectrin in membrane-related motile and anchorage functions is discussed.

L5 ANSWER 28 OF 36 MEDLINE on STN DUPLICATE 15
AN 85213864 MEDLINE
DN PubMed ID: 3923367
TI Synapsin I is a spectrin-binding protein immunologically related to erythrocyte protein 4.1.
AU Baines A J; Bennett V
NC KO4 AM00926 (NIADDK)
RO1 AM29808 (NIADDK)
RO1 GM33996 (NIGMS)
SO Nature, (1985 May 30-Jun 5) 315 (6018) 410-3.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198507
ED Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19850724
AB The membrane-associated cytoskeleton is considered to be the apparatus by which cells regulate the properties of their plasma membranes, although recent evidence has indicated additional roles for the proteins of this structure, including an involvement in intracellular transport and exocytosis (see refs 1-3 for review). Of the membrane skeletal proteins, to date only spectrin (**fodrin**) and ankyrin have been purified and characterized from non-erythroid sources. Protein 4.1 in the red cell is a spectrin-binding protein that enhances the binding of spectrin to actin and can apparently bind to at least one transmembrane protein Immunoreactive forms of 4.1 have been detected in several cell types, including brain. Here we report the purification of brain 4.1 on the basis of its cross-reactivity with erythrocyte 4.1 and spectrin-binding activity. We further show that brain 4.1 is identical to the **synaptic** vesicle protein, synapsin I, one of the brain's major substrates for cyclic AMP and Ca2+-calmodulin-dependent kinases. Spectrin and synapsin are present in brain homogenates in an approximately 1:1 molar ratio. Although synapsin I has been implicated in **synaptic** transmission, no activity has been previously ascribed to it.

L5 ANSWER 29 OF 36 MEDLINE on STN DUPLICATE 16
AN 84196409 MEDLINE
DN PubMed ID: 6144182
TI The biochemistry of memory: a new and specific hypothesis.
AU Lynch G; Baudry M
NC AG 00538 (NIA)
MH 19793-12 (NIMH)
NH 00358-03
SO Science, (1984 Jun 8) 224 (4653) 1057-63.
Journal code: 0404511. ISSN: 0036-8075.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198406
ED Entered STN: 19900319
Last Updated on STN: 20000303
Entered Medline: 19840621
AB Recent studies have uncovered a **synaptic** process with properties required for an intermediate step in memory storage. Calcium rapidly and irreversibly increases the number of receptors for glutamate (a probable neurotransmitter) in forebrain **synaptic** membranes by activating a proteinase (calpain) that degrades **fodrin**, a spectrin-like protein. This process provides a means through which physiological activity could produce long-lasting changes in **synaptic** chemistry and ultrastructure. Since the process is only poorly represented in the brain stem, it is hypothesized to be responsible for those forms of memory localized in the telencephalon.

L5 ANSWER 30 OF 36 MEDLINE on STN DUPLICATE 17
AN 84113667 MEDLINE
DN PubMed ID: 6693886
TI Evidence that a cerebellum-enriched, **synaptic** junction glycoprotein is related to **fodrin** and resists extraction with triton in a calcium-dependent manner.
AU Groswald D E; Kelly P T
NC NS 00605 (NINDS)
NS 15554 (NINDS)
SO Journal of neurochemistry, (1984 Feb) 42 (2) 534-46.
Journal code: 2985190R. ISSN: 0022-3042.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198402
ED Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19840229
TI Evidence that a cerebellum-enriched, **synaptic** junction glycoprotein is related to **fodrin** and resists extraction with triton in a calcium-dependent manner.
AB Subcellular fractions from rat cerebellum and other tissues were examined for the presence of a 240K glycoprotein, designated GP-A. Previous results have shown that GP-A is enriched in cerebellum **synaptic** junction (SJ) fractions when compared to parent **synaptic** plasma membrane (SPM) fractions and is not detected in forebrain SPM or SJ fractions. In the present studies, GP-A was not detected in myelin, mitochondria, purified nuclei, or cytosolic fractions from cerebellum, but was present in microsomal fractions. GP-A is partially soluble in the non-ionic detergent Triton X-100 and is completely soluble when cerebellum SPMs are treated with the ionic detergent N-lauryl sarcosinate. The solubilization of GP-A from cerebellum membranes was shown to be a function of bound calcium ions, e.g., pretreating SPMs with 100 microM-1mM Ca2+ decreased the solubility of GP-A in Triton by approximately threefold. GP-A is a major concanavalin A (Con A)-binding glycoprotein in cerebellum SJ fractions and migrates on sodium dodecyl sulfate (SDS) gels with a slower relative mobility than the 235K/230K **fodrin** doublet. Comparisons between purified **fodrin** and the 235K/230K doublet in cerebellum and forebrain **synaptic** fractions by two-dimensional peptide mapping indicated that they were identical. The Con A-binding property of GP-A was exploited to purify it by affinity chromatography with agarose-Con A. Peptide mapping comparisons between affinity-purified GP-A and GP-A in SPM and SJ fractions indicated that GP-A in **synaptic** fractions is apparently homogeneous. Peptide map comparisons between GP-A and 235K **fodrin** poly-peptide indicated that these two **synaptic** components are highly related (50% of their respective peptides are shared). The 235K **fodrin** polypeptide in SJs reacted with anti-**fodrin** antisera on Western blots; however, GP-A failed to cross-react. These observations, together

with results from previous studies, indicate that GP-A is highly enriched in cerebellum compared to other neuronal and nonneural tissues. Moreover, GP-A is enriched in SJs relative to SPM fractions, is related to **fodrin**, and is most likely a cell-surface glycoprotein at asymmetric synapses in cerebellum. GP-A may be involved in neuronal recognition or **synaptic** transmission in the cerebellum. The important role of calcium in **synaptic** transmission, together with the decreased solubility of GP-A in Triton that results from micromolar concentrations of calcium, suggest that GP-A may play a role in stabilizing cerebellar **synaptic** junctions.

L5 ANSWER 31 OF 36 MEDLINE on STN DUPLICATE 18
AN 84113636 MEDLINE
DN PubMed ID: 6319596
TI Calmodulin binding proteins of the cholinergic electromotor synapse: synaptosomes, synaptic vesicles, receptor-enriched membranes, and cytoskeleton.
AU Walker J H; Stadler H; Witzemann V.
SO Journal of neurochemistry, (1984 Feb) 42 (2) 314-20.
Journal code: 2985190R. ISSN: 0022-3042.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198402
ED Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19840229
AB Calmodulin binding proteins (CBPs) have been identified using a gel overlay technique for fractions isolated from Torpedo electromotor nerve endings. Different fractions possessed characteristic patterns of CBPs. Synaptosomes showed five major CBPs--Mr 220,000, 160,000, 125,000, 55,000, and 51,000. Polypeptides of Mr 55,000 and 51,000 were found in the cytoplasm and the others are membrane-associated. The Triton X-100-insoluble cytoskeleton of synaptosomes was isolated in the presence or absence of calcium. The major CBPs had Mr of 19,000, 18,000, and 16,000. In the presence of calcium, no other CBPs were seen. In the absence of calcium, an Mr 160,000 polypeptide was present in the Triton cytoskeleton. **Synaptic** vesicles showed CBPs of Mr 160,000, 25,000, and 20,000. Membrane fragments enriched in acetylcholine receptors contained two major CBPs, Mr 160,000 and 125,000, together with a less prominent protein at Mr 26,000. A protein of Mr similar to that of **fodrin** was present in synaptosomes and acetylcholine receptor membrane fragments, but only in small amounts relative to the other polypeptides observed. The heavy and light chains of clathrin-coated vesicles from pig brain did not bind calmodulin, although strong labelling of an Mr 47,000 polypeptide was found. Results showed that calelectrin does not bind calmodulin. The possible identity of the calmodulin binding proteins is discussed.

L5 ANSWER 32 OF 36 MEDLINE on STN DUPLICATE 19
AN 84044769 MEDLINE
DN PubMed ID: 6356364
TI Erythrocyte form of spectrin in cerebellum: appearance at a specific stage in the terminal differentiation of neurons.
AU Lazarides E; Nelson W J
NC GM-06965 (NIGMS)
SO Science, (1983 Nov 25) 222 (4626) 931-3.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198312
ED Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831217
AB The developing chicken cerebellum contains two forms of the plasma membrane-associated actin-binding protein spectrin. The brain form, alpha

gamma-spectrin (**fodrin**), is expressed constitutively in all neuronal cell bodies and processes during all stages of cerebellar morphogenesis. On the other hand, the erythrocyte form, alpha beta'beta-spectrin, accumulates exclusively at the plasma membrane of the cell bodies of Purkinje and granule cells and of neurons in cerebellar nuclei, but only after these cells have become postmitotic and have completed their migration to their final positions in the cerebellum. The appearance of alpha beta'beta-spectrin coincides temporally with the establishment of axosomatic contacts on these three neuronal cell types, which suggests that alpha beta'beta-spectrin accumulates in response to the formation of functional **synaptic** connections during cerebellar ontogeny.

L5 ANSWER 33 OF 36 MEDLINE on STN DUPLICATE 20
AN 83161278 MEDLINE
DN PubMed ID: 6833363
TI Identification of fodrin as a major calmodulin-binding protein in postsynaptic density preparations.
AU Carlin R K; Bartelt D C; Siekevitz P
NC 5-F32-NS06005 (NINDS)
NS 12726 (NINDS)
SO Journal of cell biology, (1983 Feb) 96 (2) 443-8.
Journal code: 0375356. ISSN: 0021-9525.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198305
ED Entered STN: 19900318
Last Updated on STN: 19970203
Entered Medline: 19830505
AB A major protein of postsynaptic densities (PSDs), a doublet of 230,000 and 235,000 Mr that becomes enriched in PSDs after treatment of **synaptic** membranes with 0.5% Triton X-100, has been found to be identical to **fodrin** (Levine, J., and M. Willard, 1981, J. Cell Biol. 90:631) by the following criteria. The upper bands of the PSD doublet and purified **fodrin** (alpha-**fodrin**) were found to be identical since both bands (a) co-migrated on SDS gels, (b) reacted with antifodrin, (c) bound calmodulin, and (d) had identical peptide maps after Staphylococcus aureus protease digestion. The lower bands of the PSD doublet and of purified **fodrin** (beta-**fodrin**) were found to be identical since both bands co-migrated on SDS gels and both had identical peptide maps after S. aureus protease digestion. The binding of calmodulin to alpha-**fodrin** was confirmed by cross-linking azido-125I-calmodulin to **fodrin** before running the protein on SDS gels. No binding of calmodulin to beta-**fodrin** was observed with either the gel overlay or azido-calmodulin techniques. A second calmodulin binding protein in the PSD has been found to be the proteolytic product of alpha-**fodrin.** This band (140,000 Mr), which can be created by treating **fodrin** with chymotrypsin, both binds calmodulin and reacts with antifodrin.

L5 ANSWER 34 OF 36 MEDLINE on STN DUPLICATE 21
AN 83262176 MEDLINE
DN PubMed ID: 6307724
TI Regulation by calcium ions of glutamate receptor binding in hippocampal slices.
AU Baudry M; Siman R; Smith E K; Lynch G
SO European journal of pharmacology, (1983 Jun 3) 90 (2-3) 161-8.
Journal code: 1254354. ISSN: 0014-2999.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198309
ED Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19830923
AB Hippocampal slices were incubated in a Krebs-bicarbonate buffer with

various concentrations of calcium and [3H]glutamate receptor binding was measured in crude **synaptic** membranes derived from these slices. Increasing the calcium concentration from 0 to 2.5 mM resulted in a 2.2-fold increase in the maximal number of the Na-independent [3H]glutamate binding sites without changes in their affinity for [3H]glutamate. This effect was totally blocked by the addition of the protease inhibitor leupeptin (50 microM) to the slice incubation medium. No effect was observed on the Na-dependent [3H]glutamate binding nor on the Na-independent [3H]glutamate binding measured in the presence of a concentration of calcium of 250 microM. Increasing the calcium concentration also resulted in an increased proteolytic activity which was inhibited by about 70% by the addition of leupeptin. Finally, increasing the calcium concentration induced the degradation of high-molecular weight proteins, the microtubule-associated proteins (MAPs) and the 220 000 dalton doublet protein corresponding to **fodrin.** Both effects were partially prevented by the addition of leupeptin in the slice incubation medium. These results indicate that the same calcium-dependent processes which were previously shown to regulate [3H]glutamate receptor binding to hippocampal membranes occur in the hippocampal slice preparation, and they suggest a mechanism by which fluctuations in calcium levels can activate a calcium-dependent proteinase, the degradation of cytoskeletal-associated proteins and the unmasking of additional glutamate receptors. The participation of such processes in various forms of plasticity is discussed.

L5 ANSWER 35 OF 36 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 1983:13090 HCAPLUS
DN 98:13090
TI Calcium(2+)- and calmodulin-dependent phosphorylation of calspectin (spectrin-like calmodulin-binding protein; fodrin) by protein kinase systems in synaptosomal cytosol and membranes
AU Sobue, Kenji; Kanda, Keiko; Yamagami, Keiji; Kakiuchi, Shiro
CS Med. Sch., Osaka Univ., Osaka, 530, Japan
SO Biomedical Research (1982), 3(5), 561-70
CODEN: BRESD5; ISSN: 0388-6107
DT Journal
LA English
AB A spectrinlike calmodulin-binding protein, calspectin (also designated as **fodrin**), in brain homogenate is concentrated in a synaptosome fraction where most (>80%) of the calspectin is associated with **synaptic** membranes. The **synaptic** membrane fraction contains an endogenous Ca2+- and calmodulin-dependent protein kinase system that catalyzes phosphorylation of the 235,000-dalton β-subunit of intrinsic calspectin. The concns. of Ca2+ and calmodulin required for the half maximal activation of the phosphorylation reaction were 1.0 and 0.8 μM, resp. The synaptosomal cytosolic fraction contained another protein kinase activity that phosphorylates both α- (mol. weight, 240,000) and β-subunits of calspectin in the presence of Ca2+ and calmodulin. The half-maximal effective Ca2+ concentration in the presence of calmodulin was 0.75 μM. The synaptosomal cytosol contained a 3rd type of calspectin kinase activity, which is independent of Ca2+ and calmodulin. Addition of cAMP (10 μM) and theophylline did not influence this activity.

L5 ANSWER 36 OF 36 MEDLINE on STN DUPLICATE 22
AN 83132254 MEDLINE
DN PubMed ID: 7160470
TI Solubilization and partial purification of protein kinase systems from brain membranes that phosphorylate calspectin. A spectrin-like calmodulin-binding protein (fodrin).
AU Sobue K; Kanda K; Kakiuchi S
SO FEBS letters, (1982 Dec 13) 150 (1) 185-90.
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198304
ED Entered STN: 19900318

Last Updated on STN: 19900318
Entered Medline: 19830407

AB In brain tissue a spectrin-like calmodulin-binding protein calspectin, or **fodrin**, is concentrated in a synaptosome fraction, where most of the calspectin is associated with the **synaptic** membranes. This endogenous calspectin was phosphorylated by protein kinase system(s) associated with the membranes. Here, we report the solubilization and partial purification of the membrane-associated calspectin kinase activity. The activity was resolved on a gel filtration column into two fractions, peaks I and II having estimated Mr of 800 000 and 88 000. The activity of peak I was dependent on the presence of both Ca2+ and calmodulin. Peak II revealed a basal activity in the absence of Ca2+ and calmodulin, which was stimulated 2-fold by addition of Ca2+. Calmodulin had no effect on the peak II activity.

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             0 SYNAPTIC
L6           0 FODRIN(P)SYNAPTIC

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FULL ESTIMATED COST                                     0.18      64.18

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CA SUBSCRIBER PRICE                                     0.00      -4.38

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=> s ipf(P)glutamate
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L7 17 IPF(P) GLUTAMATE

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L8 5 DUP REM L7 (12 DUPLICATES REMOVED)

=> d bib, hit 1-
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L8 ANSWER 1 OF 5 MEDLINE on STN DUPLICATE 1
AN 2002490847 MEDLINE
DN PubMed ID: 12350384
TI Aberrant reduction of an inhibitory protein factor in a rat epileptic model.
AU Amano Taku; Matsubayashi Hiroaki; Ozkan Eric D; Sasa Masashi; Serikawa Tadao; Ueda Tetsufumi
CS Mental Health Research Institute, Medical School, The University of Michigan, Ann Arbor, MI 48109-0669, USA.
NC NS 26884 (NINDS)
SO Epilepsy research, (2002 Sep) 51 (1-2) 81-91.
Journal code: 8703089. ISSN: 0920-1211.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200304
ED Entered STN: 20020928
Last Updated on STN: 20030424
Entered Medline: 20030423
AB Certain forms of seizure involve excessive **glutamate** transmission. We have recently identified a protein, referred to as the inhibitory protein factor (**IPF**), which potently inhibits **glutamate** uptake into isolated synaptic vesicles. In an effort to understand the mechanism underlying excessive **glutamate** transmission associated with seizure, we have analyzed **IPF** content in various brain regions of the spontaneously epileptic rat, SER (tm/tm, zi/zi), the absence-seizure tremor rat, TM (tm/tm), and the seizure-free control rats zitter ZI (zi/zi) and Wistar tremor control, each at 13 weeks of age. **IPF** content was found to be markedly reduced in the hippocampus, but not in the other brain regions, of SER, compared to the control and TM rats. TM rats also exhibited reduced **IPF** content compared to seizure-free controls. These changes appear developmentally regulated; no such alteration was observed in 8-week-old rats, which rarely show seizure. These observations indicate that an aberrant decrease in **IPF** is associated with certain forms of seizure; this decrease could lead to an abnormal increase in the amount of exocytotically released **glutamate** through its excessive accumulation in synaptic vesicles.

L8 ANSWER 2 OF 5 MEDLINE on STN DUPLICATE 2
AN 2001158843 MEDLINE
DN PubMed ID: 11181835
TI **IPF**, a vesicular uptake inhibitory protein factor, can reduce the Ca(2+)-dependent, evoked release of **glutamate**, GABA and serotonin.
AU Tamura Y; Ozkan E D; Bole D G; Ueda T
CS Mental Health Research Institute, The University of Michigan, Ann Arbor, Michigan, USA.
NC MH 15794-18 (NIMH)
NS 26884 (NINDS)
NS 36656 (NINDS)
SO Journal of neurochemistry, (2001 Feb) 76 (4) 1153-64.
Journal code: 2985190R. ISSN: 0022-3042.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 200103
ED Entered STN: 20010404
Last Updated on STN: 20010404
Entered Medline: 20010322
TI **IPF**, a vesicular uptake inhibitory protein factor, can reduce the Ca(2+)-dependent, evoked release of **glutamate**, GABA and serotonin.
AB Synaptic vesicles in the nerve terminal play a pivotal role in neurotransmission. Neurotransmitter accumulation into synaptic vesicles is catalyzed by distinct vesicular transporters, harnessing an electrochemical proton gradient generated by V-type proton-pump ATPase. However, little is known about regulation of the transmitter pool size, particularly in regard to amino acid neurotransmitters. We previously provided evidence for the existence of a potent endogenous inhibitory protein factor (**IPF**), which causes reduction of **glutamate** and GABA accumulation into isolated, purified synaptic vesicles. In this study we demonstrate that **IPF** is concentrated most in the synaptosomal cytosol fraction and that, when introduced into the synaptosome, it leads to a decrease in calcium-dependent exocytotic (but not calcium-independent) release of **glutamate** in a concentration-dependent manner. In contrast, alpha-fodrin (non-erythroid spectrin), which is structurally related to **IPF** and thought to serve as the precursor for **IPF**, is devoid of such inhibitory activity. Intrasynaptosomal **IPF** also caused reduction in exocytotic release of GABA and the monoamine neurotransmitter serotonin. Whether **IPF** affects vesicular storage of multiple neurotransmitters in vivo would depend upon the localization of **IPF**. These results raise the possibility that **IPF** may modulate synaptic transmission by acting as a quantal size regulator of one or more neurotransmitters.

L8 ANSWER 3 OF 5 HCAPLUS COPYRIGHT 2005 ACS on STN
AN 2000:699214 HCAPLUS
DN 133:286460
TI Fodrin compositions and methods for the inhibition of neurotransmitter uptake by synaptic vesicles
IN Ueda, Tetsufumi; Ozkan, Eric D.
PA Regents of the University of Michigan, USA
SO U.S., 37 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6127520	A	20001003	US 1997-840006	19970415
PRAI US 1997-840006		19970415		

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB Fodrin compns. and methods for treating neurosynaptic disorder in a subject are described. More specifically, compns. and methods for inhibiting **glutamate** uptake by synaptic vesicles in a subject are set forth. In one embodiment, the composition is inhibitory protein factor (**IPF**) and the subject is a human.

L8 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 3
AN 1998300926 MEDLINE
DN PubMed ID: 9639055
TI Glutamate transport and storage in synaptic vesicles.
AU Ozkan E D; Ueda T
CS Mental Health Research Institute, Medical School, The University of Michigan, Ann Arbor 48109, USA.
NC MH 15794-18 (NIMH)
NS 26884 (NINDS)
SO Japanese journal of pharmacology, (1998 May) 77 (1) 1-10. Ref: 47
Journal code: 2983305R. ISSN: 0021-5198.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)

(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980903
Last Updated on STN: 19980903
Entered Medline: 19980824
AB **Glutamate** plays an important metabolic role in virtually every vertebrate cell. In particular, **glutamate** is the most common excitatory neurotransmitter in the vertebrate central nervous system. As such, the mechanism by which **glutamate** is diverted from its normal metabolic activities toward its role as a neurotransmitter has, in recent years, been systematically investigated. In glutamatergic nerve endings, synaptic vesicles accumulate and store a proportion of the cellular **glutamate** pool and, in response to appropriate signals, release **glutamate** into the synaptic cleft by exocytosis. **Glutamate** accumulation is accomplished by virtue of a **glutamate** uptake system present in the synaptic vesicle membrane. The uptake system consists of a transport protein, remarkably specific for **glutamate**, and a vacuolar-type H+-ATPase, which provides the coupling between ATP hydrolysis and **glutamate** transport. The precise manner in which the **glutamate** transporter and H+-ATPase operate is currently the subject of debate. Recent data relevant to this debate are reviewed in this article. Additionally, pharmacological agents thought to specifically interact with the vesicular **glutamate** transporter are discussed. Finally, a newly discovered, endogenous inhibitor of vesicular uptake, inhibitory protein factor (**IPF**), is discussed with some speculations as to its potential role as a presynaptic modulator of neurotransmission.

L8 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 4
AN 97268710 MEDLINE
DN PubMed ID: 9108118
TI A protein factor that inhibits ATP-dependent glutamate and gamma-aminobutyric acid accumulation into synaptic vesicles: purification and initial characterization.
AU Ozkan E D; Lee F S; Ueda T
CS Mental Health Research Institute, University of Michigan, Ann Arbor 48109-0720, USA.
NC GM 07863 (NIGMS)
NS 26884 (NINDS)
SO Proceedings of the National Academy of Sciences of the United States of America, (1997 Apr 15) 94 (8) 4137-42.
Journal code: 7505876. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199705
ED Entered STN: 19970602
Last Updated on STN: 19970602
Entered Medline: 19970522
AB **Glutamate**, the major excitatory neurotransmitter in the mammalian central nervous system, is transported into and stored in synaptic vesicles. We have purified to apparent homogeneity a protein from brain cytosol that inhibits **glutamate** and gamma-aminobutyric acid uptake into synaptic vesicles and have termed this protein "inhibitory protein factor" (**IPF**). **IPF** refers to three distinct proteins with relative molecular weights of 138,000 (**IPF** alpha), 135,000 (**IPF** beta), and 132,000 (**IPF** gamma), respectively. Gel filtration and sedimentation data suggest that all three proteins share an elongated structure, identical Stokes radius (60 A), and identical sedimentation coefficient (4.3 S). Using these values and a partial specific volume of 0.716 ml/g, we determined the native molecular weight for **IPF** alpha to be 103,000. Partial sequence analysis shows that **IPF** alpha is derived from alpha fodrin, a protein implicated in several diverse cellular activities. **IPF** alpha inhibits ATP-dependent **glutamate** uptake into purified synaptic vesicles with an IC50 of approximately 26 nM, while

showing no ability to inhibit ATP-independent uptake at concentrations up to 100 nM. Moreover, **IPF** alpha inhibited neither norepinephrine uptake into chromaffin vesicles nor Na+-dependent **glutamate** uptake into synaptosomes. However, **IPF** alpha inhibited uptake of gamma-aminobutyric acid into synaptic vesicles derived from spinal cord, suggesting that inhibition may not be limited to glutamatergic systems. We propose that **IPF** could be a novel component of a presynaptic regulatory system. Such a system might modulate neurotransmitter accumulation into synaptic vesicles and thus regulate the overall efficacy of neurotransmission.

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FULL ESTIMATED COST	0.54	74.62

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CA SUBSCRIBER PRICE	0.00	-5.11

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